

AN EXPERIMENTAL ANALYSIS OF SOME METHODS AND  
MEASURES USED TO REDUCE ONE-WAY AVOIDANCE  
RESPONDING

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by

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## ABSTRACT

Two response prevention procedures were compared with a massed trials procedure for effectiveness of reducing one-way avoidance responding in rats. An important control procedure was that of matching CS-exposure duration, and hence, it was assumed, the degree of Pavlovian extinction, across the conditions. On retraining measures, the massed trials procedure was found to be superior to both the response prevention procedures which did not differ from each other. Female subjects were found to re-acquire the avoidance response more readily than male, and all subjects learned the avoidance response more readily in retraining. Of the theoretical accounts of avoidance extinction, two-process theory best accounted for the major finding of this experiment. Multivariate analyses of variance indicated that the two retraining measures were most effective in detecting experimental effects when used in combination, while the two initial training measures and the weight of the subjects were useful covariates. The two measures derived from subsequent extinction trials were of no use in detecting experiment effects.

## C H A P T E R 1

### INTRODUCTION

In the last two decades a very considerable amount of effort has been invested in research into the reduction of fear and avoidance behaviour. While interest in the classical and instrumental acquisition and extinction of these responses has existed for rather longer, the recent upsurge in interest has been concerned particularly with the investigation of procedures which facilitate response reduction (e.g. Baum, 1970), the theoretical basis of such procedures (e.g. Riccio and Silvestri, 1973), and the implications of both procedure and theory for the modification of human anxiety states and phobias (e.g. Bandura, Jeffery, and Wright, 1974; Eberle, Rehm and McBurney, 1975; Hodgson and Rachman, 1974; Sue, 1975).

One particular method of reducing the occurrence of avoidance behaviour has been at the centre of attention. The method is most commonly referred to as 'response prevention' or 'flooding' (Baum, 1971), although it has also been termed 'blocking' or 'forced reality-testing', and a variant of the procedure, used with human phobic subjects, has been termed 'implosive therapy' (Hogan, 1968, 1969; Stampfl and Levis, 1967, 1968).

Animal studies typically use a discrete-trials discriminated avoidance procedure in which the subject is first trained to a criterion of approximately three to ten consecutive avoidance responses. A trial is initiated when

the subject is in the presence of the conditioned stimulus (CS) which predicts, or is associated with the unconditioned stimulus (UCS) which is usually electric shock. The trial is terminated (as is the CS) when the subject either escapes or avoids the UCS by emitting the appropriate response - usually moving into another box, jumping onto a ledge, or pressing a bar. A shock-free inter-trial interval (approximately 30 seconds) is then given prior to the initiation of the next trial. If the response is emitted following the onset of the UCS, it terminates the UCS as well as the CS and is therefore an escape response. After several escapes the response is usually emitted within the CS-UCS interval (approximately 2 to 10 seconds), in which case it terminates the CS and prevents the occurrence of the UCS - and is therefore an avoidance response.

Response prevention is instituted once the criterion for avoidance responding has been reached. The procedure consists of turning off the shock while removing the opportunity for the subject to make the response. The subject is thereby forced to remain in the presence of the CS for (usually) a protracted period of time.

Typically, response prevention is followed by a series of extinction trials (shock-free trials) which, in all respects other than the absence of shock, are identical to the avoidance training trials. Under these conditions, it has been found that the occurrence of the response is reduced to a criterion of non-responding in fewer trials for those subjects given response prevention, than for those given normal extinction trials (Baum, 1966; Black, 1958; Carlson and

Black, 1959; Coulter, Riccio, and Page, 1969; Linton, Riccio, Rohrbaugh, and Page, 1970; Page and Hall, 1953).

Various factors have been investigated which facilitate the action of response prevention when programmed to occur during the response prevention period. Briefly, these factors are: the presence of other, non-fearful subjects (Baum, 1969c), physically forcing the subject to move around the apparatus (Lederhendler and Baum, 1970), changing illumination conditions (Baum, 1972), introducing a loud buzzer (Baum and Gordon, 1970), presenting positive intracranial stimulation (Gordon and Baum, 1971), and the introduction of nesting materials for female subjects (Reynierse and Straw, 1974).

More importantly, for present purposes, variables which determine the efficacy of response prevention have been investigated by Baum and his associates and are summarised in his 1970 article. Briefly, these variables are: the extent to which the avoidance response is trained or overtrained, the intensity of the shock used in training the avoidance response, the amount of shock trauma received, the duration of response prevention, and whether the acquisition of the response was massed or distributed.

With respect to the duration of response prevention, various studies have demonstrated that the effectiveness of the procedure increases with increasing periods of response prevention. Baum (1969b), for example, trained rats using 0.5 milliamperes shock, then gave them 0, 1, 3, or 5 minutes of response prevention. Those given one minute of response prevention exhibited virtually no reduction of the avoidance response, whereas those given 3- and 5-minute durations

showed an equal and rapid reduction of responding. When rats were trained using more intense shock (1-3 milliamperes; Baum, 1969c), and were then given 5 or 30 minutes of response prevention, only the longer duration was effective in reducing the occurrence of the avoidance response. Similar results were obtained by Coulter, Riccio and Page (1969), and by Weinberger (1965), although in the latter study response prevention was imposed in an intermittent manner rather than in a single block.

It is therefore clear that the duration of response prevention is a critical variable in determining the effectiveness of the procedure.

One theory which may account for the effectiveness of response prevention of longer durations is 'relaxation theory', advocated by Denny (1971). His main argument is that the subject begins to relax about 25 to 40 seconds post CS or UCS, the minimal optimal duration being 150 seconds. As Baum (1970) described it, "The relaxation analysis suggests that during response prevention the animal neither undergoes Pavlovian extinction of fear nor does it acquire a specific competing response; rather it learns to relax". The longer the duration of response prevention, therefore, the more effective it should be because it permits relaxation.

Data supporting the relaxation hypothesis was obtained by Baum (1969b) in an observational study of the behaviour of rats during the course of response prevention. His study demonstrated that as the occurrence of abortive avoidance behaviours (attempts to perform the avoidance response) decreased, they were gradually replaced (at around 160 seconds)



by undifferentiated general activity which could reasonably be labelled 'relaxation'.

A second explanation for the importance of longer durations of response prevention may be derived from two-process theory (Baum, 1967, 1969a; Mowrer, 1951; Riccio and Silvestri, 1973; Solomon and Wynne, 1953). Longer exposures to the CS in the absence of the UCS should result in greater Pavlovian extinction of classically conditioned fear. This is in turn manifested in the more rapid reduction of avoidance responding during normal extinction trials.

Since the duration of CS exposure has emerged as an important variable in determining the effectiveness of response prevention, it is now apparent that a number of experiments in the literature are methodologically inadequate.

Studies assessing residual fear of the CS (using passive avoidance tests), for example, have confounded different durations of CS exposure with the different treatments being investigated. Page (1955) allotted his response prevention group 10 seconds exposure to the CS whereas his regular extinction group received up to 60 seconds exposure to the CS. Coulter, Riccio and Page (1969) similarly confounded different durations of CS exposure with different experimental groups; at worst, 10 seconds exposure to the CS during response prevention was compared with up to 90 seconds CS exposure during regular extinction. In both studies therefore, there existed a clear basis for the significantly greater loss of CS aversiveness obtained for the normally extinguished animals.

This point was recognised by Bersh and Paynter (1972) who reported an experiment in which they dispensed with extinction trials in order not to confound CS exposure durations with the different treatment groups. In their experiment, response prevention with CS exposure was compared with response prevention without CS exposure, (and with other control groups), and it was demonstrated that Pavlovian extinction of the aversive properties of the CS does occur during response prevention involving prolonged CS exposure. Pavlovian extinction must therefore be assigned a contributory role in the reduction of avoidance behaviour by response prevention.

Another study confounding CS exposure durations with experimental conditions is that by Baum and Oler (1968) in which they compared the effectiveness of massed trials (shortening of the inter-trial interval during extinction trials) and response prevention in hastening the reduction of avoidance responding. In their study, massed trials subjects were taken to a criterion of 300 seconds non-responding, so that each subject had spent a variable period responding ( $t$  seconds) and a further 300 seconds meeting the criterion. For each subject, therefore, the total time involving exposure to the CS was  $t$  plus 300 seconds. To match the treatment durations of massed trials and response prevention, Baum and Oler averaged  $t$  from all massed trials subjects, and set the duration of response prevention equal to this (142.2 seconds). However, in terms of CS exposure, the massed trials subjects had spent, on average, an additional 300 seconds, in the presence of the CS.

There are potentially a large number of measures of the effectiveness of response prevention procedures, such as the number of responses made in extinction trials, the time taken to reach the response reduction criterion, passive avoidance latencies, and measures derived from retraining of the avoidance response. The most commonly used have been the number of responses made in extinction trials, and the time taken to reach the response reduction criterion. Hence, in order to use these latter two measures in their comparison of massed trials and response prevention, Baum and Oler were obliged to have extinction trials follow the response prevention condition. In effect, therefore, massed trials subjects were compared with subjects given both response prevention and extinction treatments.

As a result of their investigation (which involved other comparisons as well as that described here), Baum and Oler (1968) concluded that the massing of trials was superior to response prevention of similar duration in the reduction of avoidance behaviour. Their conclusion holds important theoretical and applied implications. On the theoretical side, it casts doubt on the importance of relaxation and of Pavlovian extinction of CS aversiveness, in the reduction of avoidance responding. This is so because both processes have more opportunity to occur during response prevention than during massed trials. The implication for applied psychologists is that the design of a human analogue of the massed trials condition would significantly advance the treatment of phobias.

Since their conclusion, and therefore the implications of it, was based on an inadequate experimental design, a more carefully controlled comparison of the two treatment methods seemed called for. The present study aimed at making such a controlled comparison.

In order to do this the experiment involved: (a) matched CS exposure time for all treatment groups, (b) matched time spent in the 'safe' area (on the ledge), (c) a comparison of male and female performances, (d) an additional response prevention treatment in order to assess the effect of necessarily altering the CS complex slightly during response prevention by removing the ledge (when using the apparatus described by Baum, 1965), (e) the use of covariates in a multivariate analysis to correct for: any initial tendency of the subjects, prior to avoidance training, to spend time in the 'safe' area; the possible influence of differences in the weights of subjects; and differences in the number and duration of shocks received in the initial avoidance training condition, (f) controls for possible order effects and confinement effects resulting from (a) and (b).

## C H A P T E R 2

### METHOD

#### 2.1 SUBJECTS

Sixty-four experimentally naive rats of the New Zealand black and white strain were used, comprising 32 of each sex. They were housed in single sex groups of approximately nine subjects with food and water freely available. At the time of the experiment they weighed between 205 and 415 gm, and were approximately 150 days old. Eight males and eight females were randomly assigned to each of the four treatment groups.

#### 2.2 APPARATUS

The apparatus had been constructed to be as similar as possible to that used by Baum and Oler (1968) which is extensively described in Baum (1965, 1966). Briefly, it consisted of an aluminium and perspex chamber 33 cm high with a 26 x 26 cm grid floor through which scrambled electric shock could be administered (0.5 ma constant current, from a Grason-Stadler model E6070B shock generator). Into one side of the cube-shaped chamber projected a 7 cm wide safety ledge. The subject could escape or avoid shock by jumping or climbing onto the ledge, where its presence was detected by a photocell system. The safety ledge was manually retractable, and a quick retraction of the ledge resulted in the subject's falling to the grid floor. The hinged ceiling was slit parallel to the edge of the ledge so that a transparent perspex slide could be lowered in order to confine the subject

on the ledge, or to prevent its jumping from the floor onto the ledge. A system of timers, counters and switches determined the CS-UCS interval, the termination of the UCS (if presented), and the inter-trial interval, and measured the response latency on each trial. Except for a 22W lamp positioned directly over the apparatus, the room was unlit and white noise was used to mask extraneous sounds.

### 2.3 PROCEDURE

Following adaptation to the apparatus, each subject received in one continuous session: first avoidance training, administration of one of the four treatment conditions, second avoidance training, and normal extinction trials.

#### Adaptation

Fifteen minutes adaptation to the apparatus (without shock) was given the day before experimentation proper, and again the following day, immediately before the first avoidance training. For five minutes within each adaptation session the ledge was available, for another five minutes the ledge was retracted, and for the remaining five minutes the subject spent 30 seconds confined on the ledge and 4 minutes 30 seconds 'confined' on the floor (ledge present, but with the perspex slide lowered into position, making the ledge unavailable). All subjects therefore received adaptation to the various arrangements (given in random order) of the apparatus. In the two 5-minute periods during which the ledge was available, the time each subject spent on the floor was recorded. This score was averaged over the two periods and was taken as a measure of any individual differences in tendency to spend time on the floor (or, reciprocally, time

on the ledge), to be used as a covariate in the data analysis.

#### First Avoidance Training

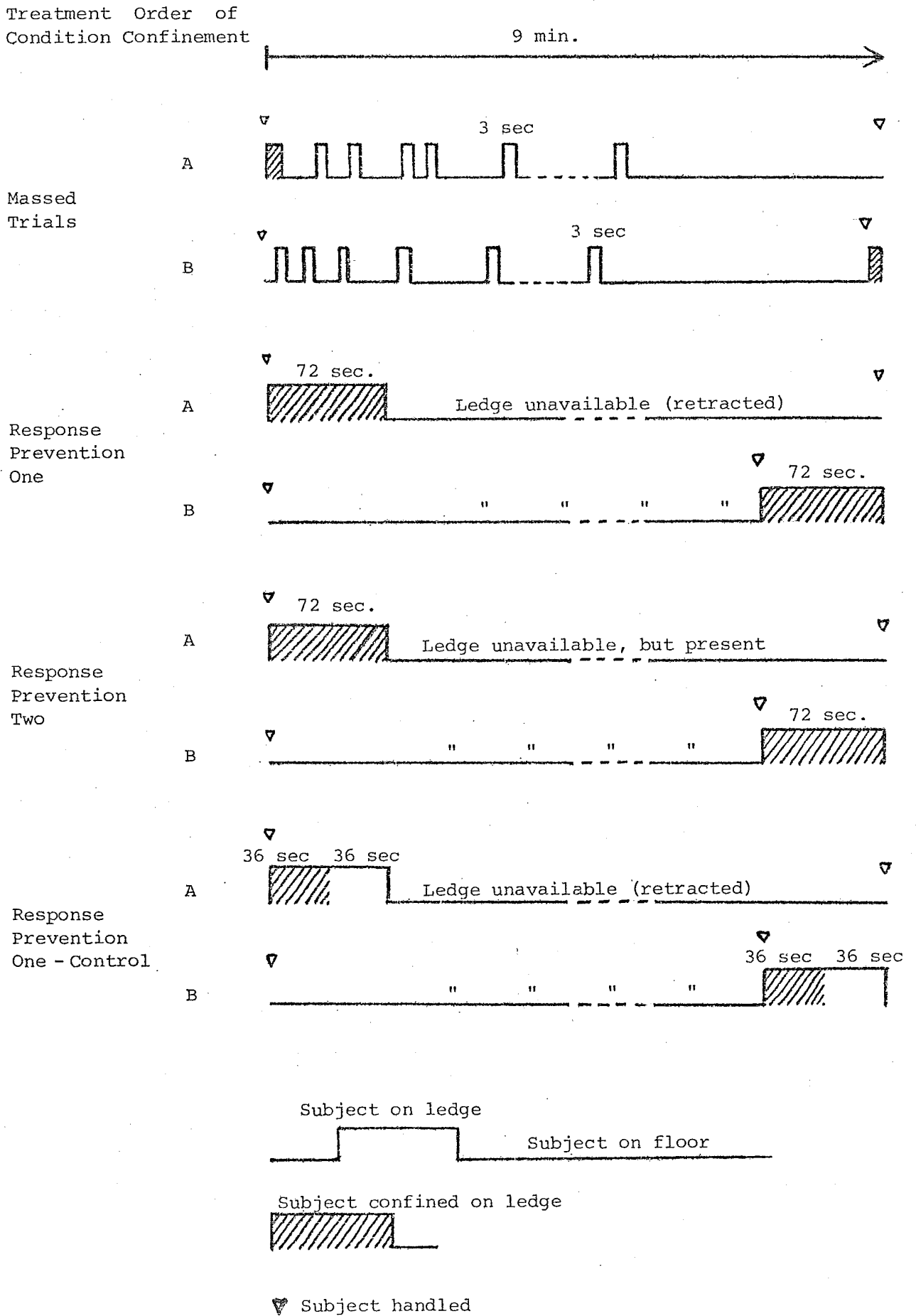
Immediately following the second session of adaptation to the apparatus, a 10 second period was given while the subject remained on the floor, then the grid was electrified and the subject received foot-shock until it escaped by jumping or climbing onto the safety ledge. The subject was allowed to remain on the ledge for 30 seconds (the inter-trial interval), after which the ledge was retracted, causing the subject to fall to the grid floor and thus initiating the next training trial. Throughout training, the subject was permitted to avoid shock by jumping or climbing onto the ledge within 10 seconds (the CS-UCS interval) of having been dropped onto the grid. The CS in this procedure was a compound stimulus combining the retraction of the ledge, the drop to the grid, and the grid floor itself with the ledge being present above the subject. Each subject was trained until it attained a learning criterion of ten consecutive avoidance responses. On each trial the response latency was recorded, from which record two measures were derived: the number of shocks received (corresponding to the number of escape responses), and the total duration of shock received ( in seconds). The number of trials required to attain the criterion was not used as a measure of learning as this was almost monotonically related to the number of escape responses.

#### Treatment

The four treatment conditions (one of which served as a control) are represented schematically in Figure 1. The total duration of treatment was nine minutes for all conditions.

FIGURE 1

SCHEMATIC REPRESENTATION OF THE TREATMENT CONDITIONS





All massed trials subjects were run first, so that the inter-trial intervals (3 seconds) could be summed and averaged to give the mean duration (72 seconds) for which massed trials subjects had been in the safe area (on the ledge). This determined the duration for which response prevention subjects were also to stay on the ledge. In this manner, safe area exposure and, hence, CS exposure durations were matched across treatment conditions. To ensure that response prevention subjects stayed in the safe area for the full 72 seconds, the perspex slide was used to confine them on the ledge. However, a control was therefore required to assess the possibly stressful effects of confinement per se. The fourth treatment group (the control) was therefore designed to be identical to the first response prevention group, with the exception that subjects were confined on the ledge for only half (i.e. 36 seconds) the normal confinement duration. In case the order of presentation of the safe-area exposure was an important influence on the effects of response prevention, half the subjects in each response prevention condition were assigned to receive order A (safe-area exposure first and response prevention second), and half to receive order B (response prevention first and safe-area exposure second). To match this for statistical purposes, massed trials subjects were assigned 5 seconds confinement on the ledge at the beginning (order A), or at the end (order B) of treatment. Occasions on which subjects had to be handled were matched for all groups as illustrated in Figure 1.

(a) *Massed trials.* Immediately after attaining the acquisition criterion, the shock-device was turned off and the subject was allowed to continue responding, but the

inter-trial interval (safe-area exposure) was reduced from 30 to 3 seconds. If the subject had been assigned to order A, it was first given 5 seconds confinement on the ledge at the beginning of massed trials, or, if it had been assigned to order B, 5 seconds ledge confinement was given following massed trials.

(b) *Response prevention one.* Having attained the acquisition criterion, and with the shock-device turned off, subjects in order A received 72 seconds confinement on the ledge (safe-area exposure). The ledge and perspex slide were then removed, to prevent the response from occurring for the remaining 7 minutes 48 seconds. Subjects in order B received the 7 minutes 48 seconds of response-prevention first, and then the 72 seconds of ledge confinement.

(c) *Response prevention two.* Subjects in this treatment condition received exactly the same procedure as those in response prevention one, with the exception that the ledge was not withdrawn during response prevention. Instead, the perspex slide was lowered thereby making the ledge unavailable to the subject. This difference from response prevention one made possible a test of the effect of necessarily altering the CS complex slightly from that present during avoidance training to that in response prevention one (where the ledge was removed) - typical of Baum's response prevention procedure.

(d) *Response prevention one control.* Subjects in this treatment condition received exactly the same procedure as those in response prevention one, with the exception that the subject was confined for only half the duration of safe-area

exposure (time on the ledge), and for the other half was free to stay there or jump down. This shorter duration of confinement served as a control to assess the possible effects of stress due to the 72 seconds of confinement in response prevention groups one and two.

#### Second Avoidance Training

At the conclusion of the treatment condition the shock-device was turned on again and avoidance training trials commenced as in the first avoidance training condition. Each subject was again trained until it attained the learning criterion of ten consecutive avoidance responses. The same measures were derived as in the first avoidance training condition.

#### Extinction Trials

Once the tenth consecutive avoidance response had been emitted in the second avoidance training condition, the shock-device was turned off but no other change in the procedure was made. Extinction trials continued until the subject met the criterion of 5 minutes without responding, or until 50 extinction trials had been completed. From this procedure two measures were derived: the number of trials required to meet the criterion, and the total number of seconds spent on the floor after 10 seconds had elapsed in each trial - a possible measure of progress made toward meeting the response reduction criterion.

## C H A P T E R    3

### RESULTS

#### 3.1 INTRODUCTION TO THE ANALYSES

Statistical analyses of the data were carried out using a multivariate analysis of variance programme (MANOVA)<sup>1</sup>. The flexibility of the programme made it ideal for this study, in that it permitted unlimited re-analyses of the data on the same run, with different variables selected to be excluded, used as covariates, or used as dependent variables.

To avoid repetitious use of cumbersome names for the design factors and variables, the abbreviations employed in the computer programme were used. These are as follows:

##### Design Factors (Independent Variables)

R : Order (ledge confinement first or last during treatment);  
A or B, respectively.

T : Treatment;

MT : Massed Trials,

RP-1 : Response Prevention 1,

RP-2 : Response Prevention 2,

RP-1C: Response Prevention 1 Control for confinement.

S : Sex of subjects;

male or female.

##### Variables (Covariates and/or Dependent Variables)

WEIGHT : The weight of subjects in grams.

TIMEONFLOR : The mean time in seconds (out of a possible 300 seconds), subjects spent on the floor when the ledge was available during the two non-shock adaptation trials.

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1. Devised by Dr Elliot Cramer at the University of North Carolina, and modified by Professor R.A.M. Gregson to run on the Burroughs computer at the University of Canterbury.

1ST SS RCD : The number of shocks subjects received during the first (i.e. pre-treatment) training of avoidance responding.

1ST DUR SS : The total duration of shock, in seconds, subjects received during the first (i.e. pre-treatment) training of avoidance responding.

2ND SS RCD : The number of shocks subjects received during the second (i.e. post-treatment) training of avoidance responding.

2ND DUR SS : The total duration of shock, in seconds, subjects received during the second (i.e. post-treatment) training of avoidance responding.

TENDTOEXT : Tendency to extinction, defined as the total number of seconds spent on the floor, counted after 10 seconds had elapsed in each trial, during the final extinction period.

The number-of-trials-to-extinction measure, taken from the final extinction period, was truncated and therefore could not be used in the statistical analyses. On this measure 35 out of the 64 subjects had not reached the extinction criterion of 300 seconds spent on the floor within 50 trials.

Seven different analyses of the data were undertaken.<sup>1</sup> The manner in which the variables were used in the analyses is summarised in Table 1.

- 
1. Prior to undertaking the analyses, a check was made of the consistency of the manual execution of the contingencies with the electrical timing of the same. This check was made for the most difficult of experimental conditions to execute (MT), and indicated that on average, 93.5% (range 88.2% to 98.7%) of the total time had been distributed appropriately. This figure was considered satisfactory, and indicated that for the other, simpler, conditions (for which insufficient data could be recorded to enable similar consistency checks), close to 100% of the total experimental time would have been correctly distributed.

TABLE 1  
SUMMARY OF THE ANALYSES

ANALYSIS	<u>UTILISATION OF VARIABLES</u>		
	EXCLUDED	COVARIATE	DEPENDENT
1			WEIGHT TIMEONFLOR 1ST SS RCD 1ST DUR SS 2ND SS RCD 2ND DUR SS TENDTOEXT
2	WEIGHT TIMEONFLOR 1ST SS RCD 1ST DUR SS		2ND SS RCD 2ND DUR SS TENDTOEXT
3		WEIGHT TIMEONFLOR 1ST SS RCD 1ST DUR SS	2ND SS RCD 2ND DUR SS TENDTOEXT
4		WEIGHT TIMEONFLOR 1ST SS RCD 1ST DUR SS 2ND SS RCD 2ND DUR SS	TENDTOEXT
5		WEIGHT TIMEONFLOR 1ST SS RCD 1ST DUR SS 2ND DUR SS TENDTOEXT	2ND SS RCD
6		WEIGHT TIMEONFLOR 1ST SS RCD 1ST DUR SS 2ND SS RCD TENDTOEXT	2ND DUR SS
7		WEIGHT TIMEONFLOR 1ST SS RCD 1ST DUR SS TENDTOEXT	2ND SS RCD 2ND DUR SS

### 3.2 ANALYSIS 1: WEIGHT, TIMEONFLOR, 1ST SS RCD, 1ST DUR SS, 2ND SS RCD, 2ND DUR SS, AND TENDTOEXT AS DEPENDENT VARIABLES

This was an exploratory analysis using all variables as dependent variables in order to provide information about their means, standard deviations, and intercorrelations.

Table 2 presents the means and standard deviations of the smallest factorial groupings (i.e. 'within-cells') for each variable. The factorial design was complete with no missing cells, and an equal number of observations (four) per cell. Larger factorial groupings of course contained multiples of four observations, resulting in up to 32 observations per cell for some tests of significance.

Table 3 presents the within-cells correlations of variables, with the standard deviation of each variable on the diagonal of the table. Only two pairs of variables were highly correlated, 1ST SS RCD with 1ST DUR SS ( $r = 0.729$ ), and 2ND SS RCD with 2ND DUR SS ( $r = 0.828$ ), indicating that during pre- and post-treatment avoidance training phases, there existed a close relation between the number of shocks, and the total duration of shock subjects received before reaching the criterion of avoidance responding. All other correlations between variables were low with the possible exceptions of the correlations between WEIGHT and 1ST DUR SS ( $r = 0.300$ ), WEIGHT and 2ND SS RCD ( $r = -0.306$ ), and WEIGHT and 2ND DUR SS ( $r = -0.469$ ). The first of these indicates that the heavier subjects tended to receive longer total shock durations during the pre-treatment avoidance training, while the latter two correlations indicate that during the post-treatment avoidance training, heavier subjects tended to receive fewer shocks and reduced total shock duration, respectively, than

TABLE 2  
WITHIN-CELLS MEANS AND STANDARD DEVIATIONS

FACTOR			VARIABLE							
R	T	S		WEIGHT	TIMEONFLOR	1ST SS RCD	1ST DUR SS	2ND SS RCD	2ND DUR SS	TENDTOEXT
A	MT	MALE	M	202.925	287.375	3.250	7.500	6.000	7.375	348.675
			SD	26.857	281.374	0.500	0.577	2.944	3.276	14.284
A	MT	FEM	M	155.975	81.500	3.500	7.500	2.750	3.625	217.950
			SD	94.951	146.152	1.291	3.136	1.500	2.136	2.987
A	RP-1	MALE	M	173.313	272.625	4.250	13.250	2.250	3.125	353.250
			SD	38.932	257.999	3.202	2.784	0.957	0.946	13.417
A	RP-1	FEM	M	177.813	80.000	4.500	9.125	1.250	1.250	219.750
			SD	43.457	154.713	1.732	3.614	0.500	0.289	13.395
A	RP-2	MALE	M	213.125	241.500	4.500	15.750	2.000	2.750	351.525
			SD	63.148	157.428	2.380	7.089	2.708	3.329	33.207
A	RP-2	FEM	M	173.500	207.875	3.250	8.000	2.250	4.000	220.200
			SD	20.740	232.306	1.258	3.082	2.062	4.378	11.420
A	RP-1C	MALE	M	177.500	323.625	4.500	10.000	1.250	1.375	334.300
			SD	54.025	236.620	2.082	4.778	0.957	0.946	21.501
A	RP-1C	FEM	M	197.188	231.250	3.500	7.500	1.250	1.250	223.125
			SD	20.985	265.120	1.000	2.273	0.957	0.866	8.540
B	MT	MALE	M	187.063	153.375	8.750	27.000	3.250	3.875	355.500
			SD	63.044	165.689	5.909	30.469	4.031	4.008	38.319
B	MT	FEM	M	187.875	85.125	4.750	9.875	4.000	5.500	222.150
			SD	46.337	143.592	0.957	3.966	1.414	2.273	6.491
B	RP-1	MALE	M	196.938	165.750	3.250	5.750	2.500	2.500	356.075
			SD	34.407	317.297	1.258	0.866	1.291	2.273	10.973
B	RP-1	FEM	M	192.563	0.000	4.000	7.625	3.000	3.500	218.750
			SD	16.126	0.000	1.414	4.211	2.160	2.915	4.489
B	RP-2	MALE	M	183.688	249.375	3.500	14.000	0.750	1.125	367.950
			SD	90.531	161.677	0.577	12.049	0.957	1.931	12.596
B	RP-2	FEM	M	170.813	171.250	5.250	11.875	1.250	1.250	221.075
			SD	39.489	200.764	3.594	1.436	0.560	0.957	11.904
B	RP-1C	MALE	M	175.125	156.875	3.250	14.125	2.500	5.875	360.525
			SD	85.074	179.393	1.256	8.635	1.915	8.230	49.859
B	RP-1C	FEM	M	166.375	257.500	5.250	13.625	2.750	2.875	215.375
			SD	20.678	182.277	2.986	11.849	1.258	1.501	10.799
COMPLETE FACTORIAL WITH NO MISSING CELLS. 4 OBSERVATIONS PER CELL.										



TABLE 3

WITHIN-CELLS CORRELATIONS OF VARIABLES WITH STANDARD DEVIATIONS ON THE DIAGONAL

VARIABLE	WEIGHT	TIMEONFLOR	1ST SS RCD	1ST DUR SS	2ND SS RCD	2ND DUR SS	TENDTOEXT
WEIGHT	20.758						
TIMEONFLOR	0.134	53.563					
1ST SS RCD	0.161	-0.126	2.384				
1ST DUR SS	0.300	-0.017	0.729	9.498			
2ND SS RCD	-0.306	0.090	-0.152	-0.117	1.879		
2ND DUR SS	-0.469	-0.070	-0.034	0.059	0.828	3.142	
TENDTOEXT	0.051	0.127	-0.211	-0.214	-0.090	-0.136	205.649

lighter subjects.

### 3.3 ANALYSIS 2: 2ND SS RCD, 2ND DUR SS, AND TENDTOEXT AS DEPENDENT VARIABLES; WEIGHT, TIMEONFLOR, 1ST SS RCD, AND 1ST DUR SS EXCLUDED

By excluding the four concomitant variables, WEIGHT, TIMEONFLOR, 1ST SS RCD, and 1ST DUR SS, from this analysis, and including them in analysis 3 as covariates, the results of the two analyses could be compared, thereby facilitating evaluation of the extent to which the four covariates refined the data analysis.

In this analysis (analysis 2), multivariate and univariate F tests were not significant at the  $p < .05$  level for both first- and second-order interactions.

For the sex main effect, multivariate and univariate F tests were similarly not significant at the  $p < .05$  level.

The treatment main effect resulted in a significant multivariate F test ( $F = 2.110$ ,  $DFHYP^1 = 9$ ,  $DFERR^2 = 112.103$ ,  $p < .034$ ) with a moderate degree of multivariate association ( $R = 0.519$ ) between independent and dependent variables. The standardised discriminant function equation for this treatment main effect was as follows:

$$V_{\text{TREATMENT}} = -1.340(2\text{ND SS RCD}) + 0.517(2\text{ND DUR SS}) \\ + 0.246(\text{TENDTOEXT}),$$

from which it is apparent that while all three dependent variables contributed to the discrimination of treatment

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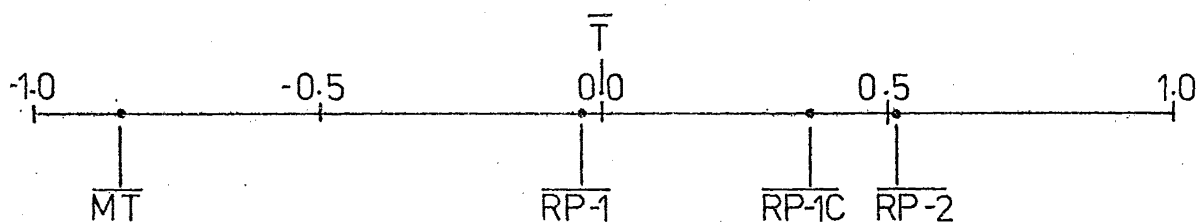
1. DFHYP: Degrees of freedom associated with hypothesis.  
2. DFERR: Degrees of freedom associated with error.

groups, the largest contributions were made by 2ND SS RCD, in particular, and 2ND DUR SS.

On the discriminant dimension used in the significant multivariate F test, the individual treatment group means ( $\overline{MT}$ ,  $\overline{RP-1}$ ,  $\overline{RP-2}$ , and  $\overline{RP-1C}$ ), are represented as deviations (-0.843, -0.033, 0.511 and 0.365, respectively) from the treatment grand mean ( $\overline{T}$ ), which is set at zero, as in Figure 2.

FIGURE 2

TREATMENT GROUP MEANS AS DEVIATIONS FROM THE TREATMENT GRAND  
MEAN IN ANALYSIS 2



From this representation several observations may be made:

(a) The means of RP-1, RP-2 and RP-1C are grouped relatively closely<sup>1</sup> together. With regard to the comparison between RP-1 and its control, RP-1C, this indicates that the confinement of subjects on the ledge during response prevention had no assessable effect on the combined three dependent variables. Likewise, in regard to the comparison between RP-1 and RP-2, the relative closeness of their means indicates that the presence or absence of the ledge during response

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1. The terms 'relatively close' and 'relatively separated' are used here, and in similar applications which follow, to indicate where the most obvious differences do or do not exist, once the multivariate F test has established a significant overall effect.

prevention had no assessable effect on the combined three dependent variables.

(b) The mean of MT is relatively well separated from the cluster of RP-1, RP-2 and RP-1C means, indicating that massed trials and response prevention treatments differed in their effects. Massed trials subjects received more shocks (2ND SS RCD) and a greater total duration of shock (2ND DUR SS) than response prevention subjects during retraining of avoidance responding. The direction of the difference on TENDTOEXT cannot be ascertained since TENDTOEXT made little contribution to the significant multivariate discrimination of treatment groups.

(c) The greatest separation between any two means is found in the discrimination between MT and RP-2. However, since the response prevention groups RP-1, RP-2 and RP-1C did not differ from each other, little importance can be attributed to the MT versus RP-2 comparison; it is merely a special case of the discrimination between massed trials and all response prevention groups, which has already been discussed in (b).

Univariate F tests on each of the three dependent variables resulted in a significant treatment main effect on 2ND SS RCD only ( $F = 5.274$ ,  $df = 3,48$ ;  $p < .003$ ), although 2ND DUR SS approached significance ( $F = 2.660$ ,  $df = 3,48$ ;  $p < .059$ ).

Multivariate and univariate F tests for the order main effect were not significant at the  $p < .05$  level.

### 3.4 ANALYSIS 3: 2ND SS RCD, 2ND DUR SS, AND TENDTOEXT, AS DEPENDENT VARIABLES; WEIGHT, TIMEONFLOR, 1ST SS RCD, AND 1ST DUR SS, AS COVARIATES

This analysis was undertaken to complement analysis 2, by employing as covariates the variables that were previously excluded.

The within-cells regression of the dependent variables on the covariates in this analysis was significant (multivariate  $F = 2.711$ ,  $DFHYP = 12$ ,  $DFERR = 111.413$ ,  $p < .003$ ;  $R = 0.644$ ). This result indicated that the dependent variables and the covariates were significantly related, and hence the adjustments made to the dependent variables removed a significant amount of covariance error.

As in analysis 2, the first- and second-order interaction effects were not significant at the  $p < .05$  level in both multivariate and univariate  $F$  tests.

In contrast to analysis 2, a significant sex main effect resulted (multivariate  $F = 7.006$ ,  $DFHYP = 3$ ,  $DFERR = 42$ ,  $p < .001$ ;  $R = 0.578$ ). For the sex effect, the standardised discriminant function equation was as follows:

$$V_{SEX} = -0.954(2ND\ SS\ RCD) + 1.679(2ND\ DUR\ SS) \\ + 0.003(TENDTOEXT),$$

from which it is apparent that the dependent variables contributing most to the male versus female discrimination were the two avoidance retraining measures, 2ND SS RCD, and 2ND DUR SS in particular, whereas the contribution made by TENDTOEXT was negligible.

The direction of the sex effect was that males received more shocks (2ND SS RCD), and a greater total duration of shock (2ND DUR SS) than females during retraining of avoidance responding. Any directional difference of male versus female performances on TENDTOEXT could not be ascertained owing to the negligible contribution that TENDTOEXT made to the sex discrimination.

Univariate F tests indicated that when the adjusted dependent variables were used individually, 2ND SS RCD resulted in a significant sex main effect ( $F = 4.992$ ,  $df = 1,44$ ;  $p < .031$ ), as did 2ND DUR SS ( $F = 16.520$ ,  $df = 1,44$ ;  $p < .001$ ), whereas TENDTOEXT did not result in a significant sex main effect at the  $p < .05$  level.

As in analysis 2, a significant treatment main effect resulted (multivariate  $F = 2.017$ ,  $DFHYP = 9$ ,  $DFERR = 102.368$ ,  $p < .045$ ) with a similar moderate degree of association,  $R = 0.530$ . The standardised discriminant function equation for this treatment main effect was as follows:

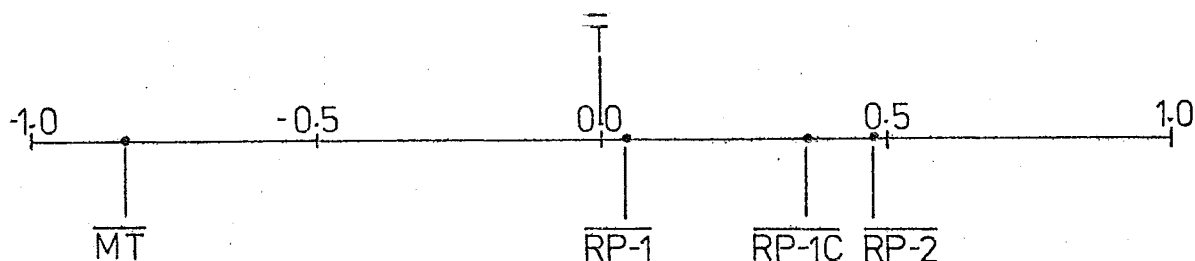
$$V_{\text{TREATMENT}} = -1.261(2\text{ND SS RCD}) + 0.350(2\text{ND DUR SS}) + 0.115(\text{TENDTOEXT}),$$

and was very similar to that for the treatment main effect of analysis 2.

On the discriminant dimension used in the significant multivariate F test, the individual treatment group means ( $\overline{MT}$ ,  $\overline{RP-1}$ ,  $\overline{RP-2}$ , and  $\overline{RP-1C}$ ) are represented as deviations ( $-0.877$ ,  $0.039$ ,  $0.477$ , and  $0.361$ , respectively) from the treatment grand mean ( $\overline{T}$ ), set at zero, as in figure 3.

FIGURE 3

TREATMENT GROUP MEANS AS DEVIATIONS FROM THE TREATMENT GRAND  
MEAN IN ANALYSIS 3.



From Figure 3, the same observations may be made as for Figure 2 relating to analysis 2, with respect to the differences between the treatment group means. However, employing covariates in this analysis resulted in the means of the three response prevention groups being closer together on the discriminant dimension than they were in analysis 2, in which covariates were not employed. In addition, the use of covariates served to increase the difference between the mean of MT and the cluster of response prevention means.

Univariate F tests on each of the three dependent variables resulted in a significant treatment main effect on both 2ND SS RCD ( $F = 5.473$ ,  $df = 3,44$ ;  $p < .003$ ) and 2ND DUR SS ( $F = 3.161$ ,  $df = 3,44$ ;  $p < .034$ ), but not on TENDTOEXT. These results, compared with those of analysis 2, indicate that the introduction of covariates into this analysis served to make 2ND DUR SS more useful in the discrimination of treatment effects.

Multivariate and univariate F tests for the order main effect were not significant at the  $p < .05$  level, as in analysis 2.

### 3.5 ANALYSIS 4: TENDTOEXT AS THE DEPENDENT VARIABLE; WEIGHT, TIMEONFLOR, 1ST SS RCD, 1ST DUR SS, 2ND SS RCD, AND 2ND DUR SS AS COVARIATES

In the preceding analyses TENDTOEXT was found to be of negligible utility as a dependent variable in both multivariate and univariate applications. Analysis 4 was therefore undertaken in order to establish whether TENDTOEXT would be of any greater utility when adjusted for the possible effects of six covariates.

Correctly named, this analysis was a univariate analysis of covariance. However, the within-cells regression of the dependent variable on the covariates indicated that TENDTOEXT and the six covariates were not significantly related at the  $p < .05$  level, and hence the adjustments made to TENDTOEXT did not remove a significant amount of covariance error.

Subsequent F tests for first- and second-order interaction effects and for sex, treatment, and order main effects were not significant at the  $p < .05$  level.

It is therefore apparent that TENDTOEXT was of virtually no utility at all as a dependent variable in this experiment.

### 3.6 ANALYSIS 5: 2ND SS RCD AS THE DEPENDENT VARIABLE; WEIGHT, TIMEONFLOR, 1ST SS RCD, 1ST DUR SS, 2ND DUR SS, AND TENDTOEXT AS COVARIATES

This univariate analysis of covariance was undertaken in order to establish how useful 2ND SS RCD was individually, as a dependent variable, when the error influence of six covariates had been statistically removed from it.



The within-cells regression indicated that the dependent variable and the six covariates were significantly related (univariate  $F = 22.232$ ,  $df = 6,42$ ;  $p < .001$ ), and hence the adjustments made to 2ND SS RCD removed a significant amount of covariance error.

The subsequent univariate  $F$  tests for main effects were non-significant at the  $p < .05$  level, although the sex main effect approached significance ( $F = 3.791$ ,  $df = 1,42$ ;  $p < .058$ ).  $F$  tests for first- and second-order interactions resulted in a significant order  $\times$  sex interaction ( $F = 4.222$ ,  $df = 1,42$ ;  $p < .046$ ). It is difficult to interpret the direction of this interaction without a full printout of the adjusted means for these groups on 2ND SS RCD; however, from the unadjusted means in Table 2 it appears likely that after order A (ledge confinement first during treatment), males received more shocks than females, but after order B (ledge confinement last during treatment), females received more shocks than males. Little weight should be given to this interpretation for the reason already mentioned, and since the interaction effect only just reached significance.

When used by itself, therefore, as a dependent variable uncontaminated by the six covariates measured in this study, 2ND SS RCD was of very little utility in detecting experimental effects.

### 3.7 ANALYSIS 6: 2ND DUR SS AS THE DEPENDENT VARIABLE; WEIGHT, TIMEONFLOR, 1ST SS RCD, 1ST DUR SS, 2ND SS RCD, AND TENDTOEXT AS COVARIATES

This univariate analysis of covariance was undertaken in order to establish how useful 2ND DUR SS was individually, as a dependent variable, when the error influence of six covariates had been statistically removed from it.

The within-cells regression indicated that the dependent variable and the six covariates were significantly related (univariate  $F = 28.915$ ,  $df = 6,42$ ;  $p < .001$ ), and hence the adjustments made to 2ND DUR SS removed a significant amount of covariance error.

Subsequent univariate  $F$  tests resulted in a significant sex main effect ( $F = 14.590$ ,  $df = 1,42$ ;  $p < .001$ ), and a significant order  $\times$  treatment  $\times$  sex interaction effect ( $F = 2.839$ ,  $df = 3,42$ ;  $p < .049$ ). For the sex difference, males received a greater total duration of shock than females. Without, however, a full printout of the adjusted means on 2ND DUR SS of the order, treatment, and sex groups, an interpretation of the direction of the significant second-order interaction was not possible. In addition, since there were only four observations per group for this interaction effect, and since the effect only just reached significance, any interpretation would, of necessity, have been tenuous.

When used by itself, therefore, as a dependent variable uncontaminated by the six covariates measured in this study, 2ND DUR SS was of little utility in detecting experimental effects. While both 2ND SS RCD and 2ND DUR SS were of more use than TENDTOEXT when employed in this manner, treatment main effects were not discriminated on any one of the three dependent variables.

### 3.8 ANALYSIS 7: 2ND SS RCD AND 2ND DUR SS AS DEPENDENT VARIABLES; WEIGHT, TIMEONFLOR, 1ST SS RCD, 1ST DUR SS, AND TENDTOEXT AS COVARIATES

The results thus far have indicated that with respect to the dependent variables, TENDTOEXT was of no utility, 2ND SS RCD and 2ND DUR SS when used together were of considerable utility (analysis 2 and, in particular, analysis 3, which employed four covariates), but when these latter two variables were employed separately, each with the other as one of six covariates (analyses 5 and 6), both became of little utility in the discrimination of experimental effects. With the additional information from analysis 1 of the high positive correlation ( $r = 0.828$ , Table 3) between 2ND SS RCD and 2ND DUR SS, it is apparent that these two variables are in fact different measures of the same underlying learning process. As such, they are complementary in the information they provide, and, for greatest utility, must be used together in any analysis.

Analysis 7 was therefore undertaken, in which only 2ND SS RCD and 2ND DUR SS were used as the dependent variables; TENDTOEXT being included with the covariates.

The within-cells regression of the dependent variables on the covariates in this analysis was significant (multivariate  $F = 3.056$ ,  $DF_{HYP} = 10$ ,  $DF_{ERR} = 84$ ,  $p < .002$ ;  $R = 0.634$ ). This result indicates that the dependent variables and the covariates were significantly related, and hence the adjustments made to the dependent variables removed a significant amount of covariance error.

Multivariate and univariate F tests were not significant at the  $p < .05$  level for both first- and second-order interaction effects.

A significant sex main effect resulted (multivariate  $F = 10.487$ ,  $DFHYP = 2$ ,  $DFERR = 42$ ,  $p < .001$ ;  $R = 0.577$ ). For this sex main effect, the standardised discriminant function equation was as follows:

$$V_{SEX} = 0.948(2ND\ SS\ RCD) - 1.674(2ND\ DUR\ SS),$$

from which it is apparent that both variables made considerable contributions to the male versus female discrimination, 2ND DUR SS making the greater contribution.

The direction of this significant sex effect was that males received more shocks (2ND SS RCD), and a greater total duration of shock (2ND DUR SS), than females during the retraining of avoidance responding. Univariate F tests indicated that the effect was significant on both of the adjusted dependent variables; for 2ND SS RCD,  $F = 4.850$ ,  $df = 1,43$ ;  $p < .033$ , and for 2ND DUR SS,  $F = 16.135$ ,  $df = 1,43$ ;  $p < .001$ .

The treatment main effect resulted in a significant multivariate F test ( $F = 2.431$ ,  $DFHYP = 6$ ,  $DFERR = 84$ ,  $p < .032$ ;  $R = 0.519$ ), for which the standardised discriminant function equation was as follows:

$$V_{TREATMENT} = 1.269(2ND\ SS\ RCD) - 0.335(2ND\ DUR\ SS).$$

From this equation it is apparent that while both variables contributed to the treatment discrimination, 2ND SS RCD made the larger contribution.

On the discriminant dimension used in the significant multivariate F test, the deviation means of the individual treatment groups, MT, RP-1, RP-2 and RP-1C, from the treatment grand mean of zero, were 0.866, -0.117, -0.450 and -0.494, respectively. Except for the change in arithmetic sign, which in itself is of no interpretable significance, these values are very close to those obtained in analysis 3 (as represented in Figure 3). Their interpretation, in terms of differences between treatment groups, is therefore the same as the interpretation made in analysis 3, with the exception that the references to TENDTOEXT made in analysis 3 do not apply to this analysis.

Multivariate and univariate F tests of the order main effect were not significant at the  $p < .05$  level.

In this analysis it was found useful to examine more closely the relative contributions of the individual covariates to the removal of error from the two dependent variables. The raw regression coefficients were therefore standardised to give two sets of beta weights, which are presented in Table 4.

TABLE 4

BETA WEIGHTS FOR THE REGRESSION OF DEPENDENT VARIABLES ON  
COVARIATES IN ANALYSIS 7

COVARIATES	DEPENDENT VARIABLES	
	2ND SS RCD	2ND DUR SS
WEIGHT	-0.001	-0.004
TIMEONFLOR	+0.000	-0.000
1ST SS RCD	-0.054	-0.134
1ST DUR SS	+0.002	+0.013
TENDTOEXT	-0.000	-0.000

From Table 4 it is apparent that of the covariates employed in this analysis, TIMEONFLOR and TENDTOEXT were of no use, relative to the remaining four covariates, in removing error from the dependent variables. WEIGHT, however, was of some use, while the two pre-treatment avoidance training measures, 1ST DUR SS, and in particular, 1ST SS RCD, were relatively the most useful in removing covariance error.

The results of this analysis precisely parallel those of analysis 3, but in this case with one less dependent variable, and with two redundant covariates.

### 3.9 SUBSIDIARY ANALYSES

Two subsidiary analyses were undertaken in order to establish whether the avoidance response was more readily learned in the second, as compared to the first training condition. It was found that subjects received fewer shocks ( $t_{\text{dep.}(63\text{df})} = 4.576, p < .001$ , two-tailed), and shorter total durations of shock ( $t_{\text{dep.}(63\text{df})} = 6.333, p < .001$ , two-tailed), in the second training condition, indicating that they learned the response more readily than in the first training condition.

## C H A P T E R 4

### DISCUSSION

#### 4.1 EFFECTS OF THE CONTROL PROCEDURES

The control group RP-1C did not differ from RP-1 (or RP-2). Therefore we may conclude that since 72 seconds of confinement produced no effect relative to 36 seconds of confinement, it is unlikely that confinement per se unduly stressed the subjects or in any way affected the results of this experiment. Ensuring the matching of safe-area exposure duration by confining the subjects on the ledge therefore appears to have been a legitimate procedure.

No difference was found between orders A or B. That is, the results were not affected by whether subjects received safe-area confinement at the beginning or at the end of the treatment.

#### 4.2 SUBSTANTIVE FINDINGS

The subsidiary analyses revealed that once the avoidance response had been learned in the first training condition, it was learned more readily in the second training condition, subjects requiring fewer shocks and shorter total durations of shock. This result confirms the same subsidiary finding made in Baum and Oler (1968), and was apparent regardless of the intervening treatment conditions designed to reduce the occurrence of avoidance responding. The implication of this finding is that the phobic person who has had his avoidance behaviour modified is still likely to relearn the response very readily given the early recurrence of appropriate learning

conditions.

Within this overall change in rate of acquisition of the avoidance response, there were clear differences in performances due to the sex of subjects and to the different treatment conditions they received. Male rats received more shocks and a greater total duration of shock than female rats during retraining of avoidance responding. Males, therefore, were slower than females in learning the response. However, there was no interaction of this sex effect with the treatment effects, therefore there appears little justification in continuing the common but wasteful practice of using only female subjects.

The treatments, RP-1 and RP-2 did not differ significantly. That is, the presence or absence of the ledge during response prevention was not important to the effectiveness of the procedure. By having the ledge present, but unavailable, RP-2, in contrast with RP-1, would seem to have had a more complete CS complex to which subjects were exposed, and hence a greater opportunity for Pavlovian extinction of fear. However, since no difference was found between RP-1 and RP-2, either the above interpretation of the procedural difference was unsound, or the ledge was an unimportant part of the CS, or the perspex slide used in RP-2 to make the ledge unavailable was a confounding influence.

Irrespective of which interpretation is correct, the lack of difference between RP-1 and RP-2 indicates that for procedural simplicity, and for consistency with previous research by Baum and his associates, response prevention with the apparatus used here is best carried out by retracting the ledge.



The massed trials treatment was found to be superior to the response prevention treatment in that relatively, it slowed the relearning of the response. Subjects which were in the massed trials group received more shocks and greater total durations of shock than subjects in the response prevention groups.

The major result of this experiment, therefore confirms Baum and Oler's (1968) conclusion: that the massing of extinction trials is more effective than response prevention in the reduction of avoidance behaviour. This holds true even when CS exposure duration is held constant across treatments as in this experiment.

The generality of this finding, while of relevance to human studies, is, strictly speaking, limited to situations comparable to those of Baum and Oler and the present situation, in which treatment continued up to approximately 8 minutes duration, and the subjects used were rats. It is appropriate here to note that in another aspect of their investigation, Baum and Oler (1968) found response prevention of 15 minutes duration to be as effective as the much shorter duration massed trials treatment. Insofar as the present finding is of relevance to applied psychologists, it suggests that some consideration be given to the design of appropriate massed trials procedures for human phobic subjects. Such a task would be difficult and applicable only in limited cases, but nevertheless potentially useful.

The superiority of massed trials to response prevention raises difficulties for current theoretical approaches. For example, this result is contrary to the result one might have

expected as a consequence of Denny's (1971) relaxation theory. The 3 seconds safe-area exposure per trial given in the massed trials condition was too brief to allow for any relaxation to have occurred there, hence little relaxation from this source could be expected to have "back-chained", or generalised to the CS (Delprato and Dreilinger, 1974; Denny, 1971) to thereby facilitate the efficacy of massed trials. Similarly, since subjects spent much of their time responding during massed trials, it seems unlikely that relaxational responses could have developed readily while the subject was on the floor.

In contrast, however, response prevention subjects spent the equivalent 7 minutes 48 seconds on the floor unable to make the response (although a number of abortive attempts to respond were made). Since Baum (1969b) has demonstrated that relaxational responses are gradually emitted from approximately 160 seconds onward during response prevention, it might be expected that relaxational responses would have been well established during the longer response prevention period used in this study. Therefore, if the learning of relaxational responses mediates the reduction of fear and avoidance behaviour, it is clear that response prevention, rather than massed trials, should have been the most effective treatment.

Similarly, differences in the degree of Pavlovian extinction of CS aversiveness occurring in the different treatment conditions cannot have been the mediating mechanism for the superiority of massed trials. If the duration of CS exposure is directly related to the degree of Pavlovian extinction of fear of the CS, then, since CS exposure

durations were matched across the different treatment conditions, the degree of Pavlovian extinction of CS aversiveness must have been the same across the conditions. This, in fact, does not necessarily pose difficulties for two-process theory as applied to the reduction of avoidance behaviour - it merely indicates that since Pavlovian extinction of CS aversiveness was held constant across the conditions, an explanation in terms of the instrumental component of two-process theory is required.

With regard to the instrumental component of the acquisition of avoidance responding, it has been cogently argued (Bolles, 1970; D'Amato, 1970; Riccio and Silvestri, 1973) that the CS also serves as a discriminative stimulus which sets the occasion for responding, and that reinforcement is provided by a reduction in shock frequency (rather than by fear reduction, as posited by Mowrer, 1951, 1960). Extending this approach to the reduction of avoidance behaviour, Riccio and Silvestri (1973 p.5) noted that, "The strong resistance to extinction of avoidance responses may well be related to the fact that traditional extinction procedures, by maintaining the CS termination contingency on successful trials, have largely ignored the discriminative function of the CS. ... The only way that S can discover the changed contingencies between CS and UCS is by failing to make the avoidance response; otherwise, he continues to receive the same sequence of events as occurred during training. In other words, the contingencies are so arranged that the discriminative aspect of the CS can only begin to weaken after the organism fails to respond..." Viewed from this perspective therefore, Riccio

and Silvestri note that response prevention may serve primarily to modify the discriminative role of the CS, since the CS is no longer experienced as being associated with or predictive of the UCS. It would be incorrect to consider modification of the discriminative role of the CS as the primary function of response prevention, since a considerable degree of Pavlovian extinction of CS aversiveness is known to take place when long enough periods of response prevention are given (Bersh and Paynter, 1972). However, since Pavlovian extinction of CS aversiveness has, theoretically, been held constant in this experiment, a closer look can be taken at possible modifications of the discriminative role of the CS in the different treatments given here.

With regard to massed trials, one change in the procedure is immediately experienced by the subject: that of the 3- instead of 30-second inter-trial interval, the result of which is to speed up the rate of responding. If it is assumed that during avoidance acquisition or normal extinction trials there is a certain probability of failures to respond, then in the speeded-up version of extinction trials (i.e. massed trials), the failures will occur earlier in the procedure and at a higher rate. Therefore it is likely that at an early stage of the procedure the subject will more frequently experience the new contingency of CS not followed by UCS. The discriminative function of the CS is therefore rapidly modified by the massing of extinction trials.

In contrast, during response prevention this repeated exposure to the new CS-UCS contingency does not occur. Compared with massed trials, which are simply speeded-up

normal extinction trials, response prevention could be said to consist of a single, but extended, normal extinction trial. The subject in response prevention therefore experiences the changed CS-UCS contingency only once. Relative to massed trials therefore, response prevention provides less opportunity for modification of the discriminative function of the CS.

Whereas response prevention is more effective than normal extinction trials in modifying the discriminative function of the CS (Riccio and Silvestri, 1973), it has become apparent that the massing of trials also modifies the discriminative function of the CS, but does so even more effectively than response prevention. Considered from the viewpoint of two-process theory this difference in the instrumental component of the procedures may well be the variable accounting for the superiority of massed trials over response prevention. To confirm this interpretation, data would necessarily have to come from detailed observational and latency records of subjects undergoing massed trials. Such data is unfortunately not available from the present study.

An alternative possibility that may account for the superiority of massed trials over response prevention is the finding (Cheng, 1966; Gaston, 1966) that the reduction of an avoidance response is a function of the magnitude of difference in effort required in learning and extinguishing it. Clearly the massing of trials during extinction required much more effort than did acquisition, hence this increase in effortfulness (compared with a decrease in effortfulness from acquisition to response prevention) may explain the finding of the present study. There is, however, no incompatibility between the response effortfulness explanation and that just expounded concerning modification of the discriminative role

of the CS. If anything, the increased effortfulness involved in a high rate of responding such as at the beginning of massed trials, would increase the probability of failures to respond within the CS-UCS interval. By increasing the probability of failures to respond and thereby causing the subject to experience the changed CS-UCS contingency, greater response effortfulness complements the previous analysis.

Competing response theory, another alternative explanation of avoidance response reduction, also requires some consideration. It should be noted that this theory derives from the experiments of Page (1955) and Coulter, Riccio, and Page (1969), in which, as has already been pointed out, the CS-exposure durations allotted to response prevention subjects were extremely short, thereby allowing little Pavlovian extinction of fear. The remaining fearfulness, it appeared to those investigators, motivated the response of freezing, which carried over from the response prevention condition to the extinction trials which followed, to 'compete' with the locomotor avoidance response. This resulted in the reduced number of responses that were made by those subjects in extinction trials, compared with subjects which had not received response prevention.

The results of these experiments by Page and associates, it would seem, can be explained more satisfactorily without invoking the notion of 'competing' responses, with the difficulties involved in testing such covert events. From two-process theory as presented here it can be seen that the high residual level of fear assessed in their response prevention subjects (and confirmed in a later replication by

Linton, Riccio, Rohrbaugh, and Page, 1970) was due to the short CS-exposure durations employed which prevented much Pavlovian extinction of CS aversiveness. With regard to the instrumental component of two-process theory, those of their subjects receiving normal extinction trials in which the CS termination contingency following a response was maintained, could not experience the changed CS-UCS contingency until they failed to respond. Hence these subjects continued to make many responses in extinction before reaching the non-response criterion, and since over this long course of extinction trials they experienced many non-shock exposures to the CS, Pavlovian extinction resulted in greatly reduced CS aversiveness for those subjects as compared with response prevention subjects.

Because their response prevention subjects could not emit the avoidance response, these subjects experienced the changed CS-UCS contingency, thereby weakening the discriminative control of the CS over avoidance responses. But because the fear eliciting properties of the CS had not been extinguished, it is reasonable to assume that another response in the hierarchy of species-specific defense reactions (Bolles, 1970) was therefore emitted with high frequency in the presence of the CS. Hence, in subsequent presentations of the CS, as in normal extinction trials, the new response (freezing) continued to be emitted. In this analysis the notion of 'competing' responses is redundant.

This two-process analysis of avoidance extinction logically extends to encompass studies in which CS-exposure is continued for longer durations. In such instances, by

Pavlovian extinction, the CS is reduced to a neutral status in terms of its fear-eliciting properties, and correspondingly 'relaxed' responses are emitted with a high frequency. When this occurs within response prevention, the CS also has its discriminative control over the avoidance response weakened, and the 'relaxed' responses already occurring with a high frequency in the presence of the (neutral) CS, are therefore those most likely to be emitted in the presence of the CS at a later occasion, such as in normal extinction trials. An observational study of behaviour occurring during response prevention of prolonged CS-exposure (Baum, 1969b) appears to support these contentions.

It therefore appears, in summary, that two-process theory in the form advocated here adequately accounts for the results of normal extinction trials, response prevention, and the present finding of the superiority of massed trials to response prevention. Increased effortfulness in responding required in extinction conditions is a complementary concept to two-process theory, while the notion of competing responses has been found redundant. Relaxation theory seems unable to account for the results of this experiment, but since observational data were not taken, it cannot be said to have been contradicted. However, relaxation theory cannot account for the results of the experiments by Page and his associates, hence is limited in comparison to two-process theory.



#### 4.3 METHODOLOGICAL FINDINGS

The use of multivariate analyses of variance on the data of this study proved particularly productive. Whereas univariate analyses on occasions did not detect experimental effects, the combination of several dependent variables in multivariate analyses did.

In particular, the number of shocks and the total durations of shock received during retraining of the avoidance response were of little use as dependent variables in the univariate situation, but when combined in the multivariate situation they proved to be of considerable utility. Aside from commonsense considerations, these two dependent variables were statistically found to be two complementary measures of the same underlying learning process - avoidance response acquisition.

The use of covariates in this study also proved to be of benefit. Several of the variables used in this study as covariates served to refine the data analysis, making obvious experimental effects which had previously been obscured by individual differences on those variables. The covariates which were of particular use were the number of shocks, and the total durations of shocks, subjects received during the initial avoidance training condition. To a lesser extent the weight of subjects was a useful covariate, while the time subjects spent on the floor during adaptation trials, a measure of possible position bias, was of no use as a covariate.

The finding of the usefulness of the two initial training measures as covariates holds implications for other studies of avoidance behaviour. The usual method of controlling

for these confounding variables has been to statistically test for significant differences between groups of subjects on these measures and to proceed to further analyses only if the groups did not differ. Clearly this is a wasteful method compared to that of using covariates.

The two measures derived from the extinction trials were of no use as dependent variables in this study. The maximum number of extinction trials given was 50, which was found to have been too few as many subjects did not reach the response reduction criterion within that limit, therefore truncating one measure - the number of trials required to attain the criterion. Tendency to extinction, possibly less sensitive to the 50 trial limit, was also found to be of little utility in detecting experimental effects. This might have been due to the effects of the second avoidance training condition reducing the differences between the experimental groups. This explanation is difficult to support, since the design factor effects were established to have been present on the measures derived from second avoidance training anyway, and these differences would therefore be expected to have persisted into the extinction trials, in the manner found by Franchina, Hauser, and Agee (1975).

An improvement which could have been made to this study would have been the inclusion of a measure of CS aversiveness. Since the design of this study provides a paradigm for holding the Pavlovian extinction of fear constant across experimental conditions (assuming that the duration of CS exposure is the sole determinant of the degree of Pavlovian extinction), a passive avoidance test following the treatment condition would have been appropriate to test this assumption.

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